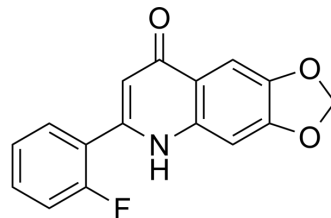


CHM-1

| | | | |
|--------------------|--|-------|----------|
| Cat. No.: | HY-103257 | | |
| CAS No.: | 154554-41-3 | | |
| Molecular Formula: | C ₁₆ H ₁₀ FNO ₃ | | |
| Molecular Weight: | 283.25 | | |
| Target: | Microtubule/Tubulin | | |
| Pathway: | Cell Cycle/DNA Damage; Cytoskeleton | | |
| Storage: | Powder | -20°C | 3 years |
| | | 4°C | 2 years |
| | In solvent | -80°C | 6 months |
| | | -20°C | 1 month |



SOLVENT & SOLUBILITY

In Vitro

DMSO : 5 mg/mL (17.65 mM; Need ultrasonic)
 H₂O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

| Concentration | Solvent | Mass | | |
|---------------------------|---------|-----------|------------|------------|
| | | 1 mg | 5 mg | 10 mg |
| Preparing Stock Solutions | 1 mM | 3.5305 mL | 17.6523 mL | 35.3045 mL |
| | 5 mM | 0.7061 mL | 3.5305 mL | 7.0609 mL |
| | 10 mM | 0.3530 mL | 1.7652 mL | 3.5305 mL |

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

CHM-1, a microtubule-destabilizing agent, inhibits tubulin polymerization. CHM-1 is a potent and selective antimetabolic antitumor activity against human hepatocellular carcinoma. CHM-1 induces growth inhibition and apoptosis via G₂-M phase arrest in human hepatocellular carcinoma cells by activation of Cdc2 kinase activity^{[1][2][3]}.

IC₅₀ & Target

IC₅₀: 0.75 μM (HA22T)^[1]

In Vitro

CHM-1 (0-100μM; 24 hours) induces significant concentration-dependent growth inhibition in HA22T, Hep3B, and HepG2 cells, with the most potent effects observed in HA22T cells (IC₅₀ = 0.75 μM)^[1].

CHM-1 (0-10 μM; 24 hours) significantly increases the binding of cyclin B1 to Cdc2 in HA22T cells^[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay^[1]

| | |
|------------|-------------------------------|
| Cell Line: | HA22T, Hep3B, and HepG2 cells |
|------------|-------------------------------|

| | | |
|----------------|--|--|
| | Concentration: | 0-100 μ M |
| | Incubation Time: | 24 hours |
| | Result: | Induced G ₂ -M arrest of the cell cycle followed by apoptosis. |
| | Western Blot Analysis ^[1] | |
| | Cell Line: | HA22T cells |
| | Concentration: | 0-10 μ M |
| | Incubation Time: | 24 hours |
| | Result: | Induced change in expressed and phosphorylated status of G ₂ -M regulators in human hepatocellular carcinoma cells. |
| In Vivo | CHM-1 (10 mg/kg; i.p.) induces a dose-dependent inhibition of HA22T tumor growth ^[1] . MCE has not independently confirmed the accuracy of these methods. They are for reference only. | |
| | Animal Model: | Male severe combined immunodeficient mice (HA22T) ^[1] |
| | Dosage: | 10 mg/kg |
| | Administration: | i.p. |
| | Result: | Induced a dose-dependent inhibition of HA22T tumor growth. |

REFERENCES

- [1]. Wang SW, et al. CHM-1, a novel synthetic quinolone with potent and selective antimitotic antitumor activity against human hepatocellular carcinoma in vitro and in vivo. *Mol Cancer Ther.* 2008 Feb;7(2):350-60.
- [2]. Liu CW, et al. CHM-1, a novel microtubule-destabilizing agent exhibits antitumor activity via inducing the expression of SIRT2 in human breast cancer cells. *Chem Biol Interact.* 2018 Jun 1;289:98-108.
- [3]. Tsai AC, et al. CHM-1, a new vascular targeting agent, induces apoptosis of human umbilical vein endothelial cells via p53-mediated death receptor 5 up-regulation. *J Biol Chem.* 2010 Feb 19;285(8):5497-506.

Caution: Product has not been fully validated for medical applications. For research use only.

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